to mention the possibility that joint rupture could be the presenting feature of inflammatory arthritis. When joint rupture complicates arthritis it is almost always restricted to early joint involvement.[1] In patients presenting with joint rupture systemic examination and the investigation should be performed to exclude this possibility. Even if no obvious cause is found at the time of presentation many patients will develop frank local synovitis or evidence of a generalised rheumatic disease within a year or two.

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[1] Jayson MIV, Swannell AJ, Kirk JA, Dixon AStJ. *Ann Phys Med* 1969-70;**10**:175-9.

SIR,—We were interested to read the report by Drs D G Macfarlane and P A Bacon of popliteal cyst rupture in normal knee joints (1 November, p 1203). This finding is, however, not entirely new. One of Baker's original eight cases[1] had no evidence of disease of the knee joint until the joint became infected after aspiration of the cyst and one other had only slight swelling of the knee since a sprain two years previously. Baker also gives the history of an army officer (originally described by Foucher in 1856) who developed a chronic knee effusion after a forced march on rough ground and who subsequently developed a popliteal cyst which ruptured. His symptoms eventually resolved and left him with an apparently normal knee.

In our own series of 43 patients presenting with symptoms suggestive of deep vein thrombosis,[2] 16 had popliteal cysts, of whom only two had rheumatoid arthritis, six had mild degenerative joint disease, and eight had no previous symptoms referable to the knee, though one of these had crepitus on passive flexion. Furthermore, five of these 16 patients had venographically proved deep vein thrombosis as well as popliteal cysts. We suggest that the presence or suspicion of popliteal cyst should not lead the clinician to conclude that a deep vein thrombosis can be excluded.

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Toxic shock and tampons

SIR,—The leading article "Toxic shock and tampons" (1 November, p 1161) stated that the syndrome is as yet unreported in Britain.

I suffered the characteristic symptoms on three separate occasions in 1978. On the first occasion I had just been fitted with a contraceptive diaphragm and was somewhat inexpert at inserting it. On the day following the first occasion of its use I developed, in a matter of hours, high fever with rigors, a centripetal erythematous rash, vomiting, diarrhoea, muscle tenderness and stiffness, backache, and severe pelvic pain. This lasted about four days and the symptoms subsided quite abruptly, leaving a residual pain in the left iliac fossa. On this occasion my GP prescribed co-trimoxazole, believing the illness to be an atypically presenting salpingitis. The second episode was 20 days after the first and again followed insertion of the diaphragm. The symptoms were the same but in addition I suffered severe vaginitis and a purulent vaginal discharge. Cultures from the swabs taken showed *Staphylococcus aureus*. The third episode occurred 16 days after the second. This time I was on holiday, menstruating and using extra-absorbent tampons (Lil-lets Super Plus). I experienced the same symptoms but they lasted barely two days.

By this time I was extremely worried and on my return home I consulted a gynaecologist, who could find no other explanation than salpingitis for my symptoms (and the continuous left iliac pain). Since then the pain has gradually disappeared; I have avoided using internal tampons (especially the highly absorbent kind) so far as possible, and greater skill in inserting the diaphragm has meant less possibility of internal trauma. On reading the reports of "toxic shock syndrome" this year,[1] I immediately recognised the cardinal signs of my mystery illness of 1978.

I think that it is important to consider that tampons need not be the only cause of "toxic shock syndrome" and suggest that inexpert use of the contraceptive diaphragm may also give rise to the condition, which can occur therefore at times other than during menstruation.

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SIR,—We read your leading article on toxic shock and tampons (1 November, p 1161) and agree with your statement that "it would be curious indeed if such a disease were to be confined to the United States." The syndrome has been described in Sweden,[1-2] and we want to report a further case with severe circulatory symptoms.

The patient was a 19-year-old woman with normal periods who used tampons of a common Swedish type. She fell ill on the fourth day of her period, with pains in her body and joints, severe headache, and high fever, followed by severe vomiting and watery diarrhoea. Because of rapid deterioration she was brought to hospital 36 hours after the onset of her illness. On admission she was in shock; her blood pressure was 70/55 mm Hg; she had a tachycardia with a pulse rate of 150 beats/min; and her temperature was 40·6°C. Bilateral conjunctivitis was noted but there were no exanthema.

The patient had a foul-smelling discharge from the vagina, and *Staphylococcus aureus* of phage group I and *Escherichia coli* were grown from vaginal secretions taken from the tampon. The patient was treated with intravenous fluids, plasma, methylprednisolone and cefuroxime. An improvement in central as well as peripheral circulation occurred, urine production started, and the central venous pressure rose to 8·5 cm water within three hours of treatment. After 12 hours of treatment, however, the blood pressure as well as the central venous pressure fell, diuresis stopped, and the patient became disoriented. Dopamine hydrochloride and digitalis were added to the previous treatment and produced a prompt improvement in her circulation and diuresis. Two days later, however, her heart was dilated, and she had symptoms of pulmonary stasis, but no pericardial effusion could be seen. An extreme sinus arrhythmia with pulse rates down to 35 beats/min was noticed. The decompensation was treated symptomatically, and the symptoms slowly disappeared. A week after starting treatment all signs of cardiovascular problems had disappeared.

Cultures from the blood, cerebrospinal fluid, and urine were negative at admission. *Staph aureus* of phage group I was cultured from the nasopharynx as well as from the tampon. On the third day the patient had a typical strawberry tongue, and within a fortnight intense desquamation of the palms occurred. But she never developed any exanthema during the course of the disease.

This patient fulfils the criteria for the toxic shock syndrome (as reported by Daley *et al* and Davis *et al* at the Interscience Conference on Antimicrobial Agents and Chemotherapy, New Orleans, 1980) with the exception of the missing exanthema. She used tampons regularly and changed them frequently in the daytime, but used one tampon at night. This habit seems to be quite normal. During the previous period she had a one-day illness with high fever; and pain in the body and joints on the last day of the bleeding, but this illness cleared up spontaneously. This accords with the description of recurrence of toxic shock syndrome which occurs in about 30% of the patients.

Staph aureus of phage-group I was initially reported to be associated with toxic shock syndrome,[3] but this has not been substantiated from the United States. *E coli* is a known toxin producer. Whether finding *E coli* together with *Staph aureus* is important in the toxic shock syndrome has not been discussed.

This case, and the other reports from Sweden, show that the toxic shock syndrome is not an American problem only, but can occur in other parts of the world or at least in Scandinavia.

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Methylene blue is dangerous

SIR,—I was surprised to read the suggestion in "Any Questions" (11 October, p 981) that the intrathecal administration of methylene blue could be carried out to establish the occurrence of cerebrospinal fluid rhinorrhea.

Evans and Keegan[1] described 14 cases of neurotoxic adverse effects of intrathecal methylene blue; neurological sequelae included paraplegia, radiculopathy, cauda equina syndrome, encephalopathy, optic neuritis, and meningeal irritation. They concluded that methylene blue should not be administrated intrathecally. Schultz and Schwartz[2] described a patient who suffered extensive damage to the spinal roots and spinal cord following intrathecal methylene blue and reported an additional three cases.

The most recent report of neurological sequelae following this procedure is that of Sharr *et al*,[3] who describe a 59-year-old man who was given intrathecal methylene blue in an attempt to locate the source of cerebrospinal rhinorrhoea. He developed a progressive paraparesis with urinary retention, which progressed over 3½ years after the intrathecal

injection to a total paraplegia at the level of T9 dermatome and persisted until the patient's death five years later. The packaging insert for methylene blue injection states that intraspinal injection is contraindicated.

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[2] Schultz P, Schwarz GA. *Arch Neurol* 1970;**22**:240-4.
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Phenylbutazone overdose

SIR,—I should like to comment on the report by Dr L F Prescott and others (25 October, p 1106) of a case of phenylbutazone overdose. They state that a case of phenylbutazone poisoning apparently had not been reported previously in the United Kingdom. I should like to draw their attention to such a report.[1]

Despite what Dr Prescott and his colleagues say about phenylbutazone being extensively metabolised and highly bound to plasma proteins, it is inaccurate to state that haemoperfusion is unlikely to enhance elimination significantly. In the case quoted, we were able to show a rapid reduction of blood phenylbutazone concentrations during haemoperfusion through columns of activated charcoal (Haemocol—Smith and Nephew Pharmaceutics Ltd). The use of haemoperfusion in this particular case was almost certainly life saving. The technique would seem to be indicated in severe cases of phenylbutazone overdose.

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[1] Strong JE, Wilson J, Douglas JD, Coppel DL. *Anaesthesia* 1979;**34**:1038-40.

Primary biliary cirrhosis: an epidemiological study

SIR,—Dr David R Triger (20 September, p 772) and Dr W Stuart Hislop (18 October, p 1069) both record extensive experience with liver histology in their studies of primary biliary cirrhosis. Dr Triger says that "the diagnosis was *supported* [my italics] by histological findings in all patients who underwent biopsy, though in many cases the diagnoses were "consistent with" rather than "diagnostic of" primary biliary cirrhosis.

Dr Hislop writes, "In all cases the diagnosis was based on the well-described clinical features, and mitochondrial antibodies were uniformly present. Histological confirmation of the diagnosis was obtained in all cases"—two at necropsy. Dr Triger mentions that two elderly women were not biopsied as the procedure could not have been ethically justified. Of his 32 patients who had biopsies, histological specimens were obtained surgically on six occasions and percutaneously on 50 occasions. Only one patient (who had otherwise classical features of primary biliary cirrhosis) had no antimitochondrial antibodies.

These studies confirm the reliability of biochemical and serological data in the diagnosis of primary biliary cirrhosis.[1] If the role of biopsy is "supportive" or "confirmatory" at best, and most often merely "consistent with," then the place of histology is surely best reserved for cases where the biochemical or the serological evidence is lacking despite careful follow-up and repeated measurement.[2] If biopsy is no longer routinely necessary for diagnosis, does it contribute to management? These questions are of practical importance for those of us who wish to screen patients with rheumatic symptoms for evidence of hepatic abnormality; to identify patients with early and non-hepatic features of primary biliary cirrhosis; and to elucidate further the relationship between polymyalgia rheumatica, a common symptom complex with protean manifestations, and other diseases.

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[1] Batstone GF, Robertson JC, Loebl WY. *Br Med J* 1979;i:125-6.
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Endometriosis—continuing conundrum

SIR,—Your leading article "Endometriosis—continuing conundrum" (4 October, p 889) raises a number of interesting points. It is the experience of most gynaecologists with a major interest in infertility that this condition is far more common than previously held and if looked for is frequently diagnosed. A high index of suspicion, examination at the appropriate time of the menstrual cycle, including a rectal examination, and the use of laparoscopy all contribute to the increasing frequency with which it is diagnosed.

The dilemma that faces the gynaecologist, having diagnosed a minor degree of endometriosis, is whether per se it is a significant factor in causing the infertility. The association of endometriosis with disturbances of ovarian function needs to be established in carefully controlled investigations. However, the demonstration of endometriosis should lead the clinician into measuring more sensitive indicators of ovarian function, such as plasma progesterone levels in the luteal phase, rather than relying on basal temperature charts and secretory changes in endometrial biopsies. Once demonstrated, poor ovarian function can be rectified by treatment with antioestrogen ovarian stimulants such as clomiphene or tamoxifen.

Another possible mechanism of infertility in a woman with endometriosis is an adverse effect on survival of sperm in the pelvis. Indeed, few sperm have been found in pouch of Douglas aspirates in women with endometriosis (Hammerstein, personal communication). Survival of sperm in peritoneal fluid aspirated from the pouch of Douglas was measured by autocorrelation analysis of scattered laser light (with apparatus developed for this purpose within this department to measure sperm velocity). In aspirates obtained from the normal pelvis the survival and motility of sperm was enhanced compared with control sperm diluted in saline (n=20). In subjects with a minor degree of endometriosis no adverse effects on sperm motility and velocity were observed (n=8). However, in aspirates obtained from all women with more extensive endometriosis a major degree of inhibition of sperm survival and motility was observed (n=6).

This is an early ongoing study and more data are being collected. However, these preliminary data clearly indicate that a factor, as yet undetermined, appears to be released from significant areas of endometriosis into the peritoneal fluid, which has an adverse effect on sperm velocity and survival. It remains to be demonstrated whether medical treatment, with or without conservative surgery, can correct this apparent obstacle to fertility.

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Adverse reaction to bupivacaine

SIR,—Reporting a case of neurotoxicity following the use of bupivacaine for intravenous regional analgesia (18 October, p 1043), Dr A M Henderson quoted figures for peak plasma bupivacaine of 840 ng/ml four minutes after release of the tourniquet.[1] These were the means of measurements of systemic venous plasma levels in five subjects. Another small study measured arterial blood concentrations and found peak levels between 4900 ng/ml and 11 000 ng/ml one minute after tourniquet release, a similar technique having been used.[2] Since plasma concentrations are generally 1·6 times those of whole blood[3] there is an enormous discrepancy between the findings, which even a recognised higher concentration associated with arterial sampling[3 4] cannot explain.

Where the concentrations of bupivacaine that may follow an intravenous regional block are not predictable then toxic sequelae, although rare, should not be totally unexpected. In order to increase the safety of this technique I would suggest, firstly, that an intravenous needle or cannula be placed in an unoccluded limb prior to any injection of local anaesthetic or tourniquet release. Secondly, since the effluent blood from the arm after tourniquet release carries considerable quantities of local anaesthetic for several minutes,[2 5] reinflation of the tourniquet should be considered at the first signs of toxicity.

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McIlroy—a suggestion

SIR,—Dr Pallis and I always seem to be one jump behind McIlroy (save an episode last year when serendipity landed him in a geriatric bed under the care of my wife); but I am sure that Drs A J McGennis and M J Corry (1 November, p 1217) would like to know that, since leaving Dublin in September, McIlroy has visited Glasgow—where, it seems, he was genuinely ill—and St Mary's, Praed Street, and Guy's Hospital in London.

The needs of brevity left us unable to give any details of McIlroy's numerous admissions in our original paper, but we can confirm that he had undergone psychiatric assessment at